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Authors

Eisner, MD
Katz, PP
Yelin, EH
et al.

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Measurement of Environmental Tobacco Smoke Exposure among Adults with Asthma

Mark D. Eisner,¹ Patricia P. Katz,² Edward H. Yelin,² S. Katharine Hammond,³ and Paul D. Blanc¹

¹Division of Occupational and Environmental Medicine and Division of Pulmonary and Critical Care Medicine, Department of Medicine, and ²Institute for Health Policy Studies and Department of Medicine, University of California, San Francisco, San Francisco, California, USA; ³Center for Occupational and Environmental Health and School of Public Health, University of California, Berkeley, California, USA

Because the morbidity and mortality from adult asthma have been increasing, the identification of modifiable environmental exposures that exacerbate asthma has become a priority. Limited evidence suggests that exposure to environmental tobacco smoke (ETS) may adversely affect adults with asthma. To study the effects of ETS better, we developed a survey instrument to measure ETS exposure in a cohort of adults with asthma living in northern California, where public indoor smoking is limited. To validate this survey instrument, we used a passive badge monitor that measures actual exposure to ambient nicotine, a direct and specific measure of ETS. In this validation study, we recruited 50 subjects from an ongoing longitudinal asthma cohort study who had a positive screening question for ETS exposure or potential exposure. Each subject wore a passive nicotine badge monitor for 7 days. After the personal monitoring period, we readministered the ETS exposure survey instrument. Based on the survey, self-reported total ETS exposure duration ranged from 0 to 70 hr during the previous 7 days. Based on the upper-range boundary, bars or nightclubs (55 hr) and the home (50 hr) were the sites associated with greatest maximal self-reported exposure. As measured by the personal nicotine badge monitors, the overall median 7-day nicotine concentration was 0.03 $\mu\text{g}/\text{m}^3$ (25th–75th interquartile range 0–3.69 $\mu\text{g}/\text{m}^3$). Measured nicotine concentrations were highest among persons who reported home exposure (median 0.61 $\mu\text{g}/\text{m}^3$), followed by work exposure (0.03 $\mu\text{g}/\text{m}^3$), other (outdoor) exposure (0.025 $\mu\text{g}/\text{m}^3$), and no exposure (0 $\mu\text{g}/\text{m}^3$; $p = 0.03$). The Spearman rank correlation coefficient between self-reported ETS exposure duration and directly measured personal nicotine concentration during the same 7-day period was 0.47, supporting the survey's validity ($p = 0.0006$). Compared to persons with no measured exposure, lower-level [odds ratio (OR) 1.9; 95% confidence interval (CI), 0.4–8.8] and higher-level ETS exposures (OR 6.8; 95% CI, 1.4–32.3) were associated with increased risk of respiratory symptoms. A brief, validated survey instrument can be used to assess ETS exposure among adults with asthma, even with low levels of exposure. This instrument could be a valuable tool for studying the effect of ETS exposure on adult asthma health outcomes. **Key words:** asthma, biological markers, environmental monitoring, nicotine, smoking, tobacco smoke pollution. *Environ Health Perspect* 109:809–814 (2001). [Online 13 August 2001] <http://ehpnet1.niehs.nih.gov/docs/2001/109p809-814eisner/abstract.html>

During the past decade, the morbidity and mortality from adult asthma have been increasing. Consequently, identifying modifiable environmental factors that exacerbate asthma has become a priority. Limited evidence indicates that exposure to environmental tobacco smoke (ETS) may exacerbate asthma among adults (1–4). Further research is necessary to establish the causal connection between ETS exposure and adverse asthma health outcomes in adults. To elucidate the effects of ETS better, we developed a survey instrument to measure ETS exposure in a cohort of adults with asthma living in northern California.

Previous questionnaires have been designed to assess ETS exposure, but they have limitations for studying the effect of ETS exposure on asthma health status in adults. The validation of most previous ETS questionnaire instruments occurred in places and times at which cigarette smoking was more common. Consequently, their utility in California, where the prevalence of smoking

currently is substantially lower than in most other states, is unclear (5). Since most previous ETS survey instruments have been published, California state law has prohibited smoking in nearly all workplaces effective 1 January 1995 (6). This change may have affected the locations of ETS exposure, including a shift in indoor workplace exposure to outdoor locations. Moreover, previous instruments have not ascertained asthma-related ETS effects, such as respiratory symptoms and medication use. To study the effects of ETS on adults with asthma living in northern California, we developed a survey instrument that measures exposure in relevant microenvironments, including outdoor workplaces.

Another limitation of previous studies is validation using serum, urine, or salivary cotinine, a nicotine metabolite and biomarker of ETS exposure (7–16). Because it has substantial person-to-person variability due to uptake, metabolism, and elimination,

cotinine has limitations as a biomarker of ETS exposure (17). Cotinine is also found in several foods, reducing its specificity for ETS when exposure levels are very low (17,18). The half-life of cotinine is relatively short (1–2 days), which limits its utility for assessing exposure during a longer period. To validate the ETS exposure survey instrument, we used a passive badge monitor that measures actual exposure to ambient nicotine, a more direct and specific measure of personal ETS exposure (19,20).

Materials and Methods

Overview. We developed a survey instrument to measure ETS exposure among adults with asthma living in northern California. To validate the instrument, we recruited 50 subjects from an ongoing longitudinal asthma cohort study who had a positive screening question for ETS exposure or potential exposure. Each subject wore a passive nicotine badge monitor for 7 days. After the personal monitoring period, we readministered the ETS exposure survey instrument. The study was approved by the University of California San Francisco Committee on Human Research.

Subject recruitment. We used data collected during an ongoing prospective, longitudinal cohort study of adults with asthma recruited from physician practices in northern California. Details of recruitment have been reported previously (21–23). In brief, we initially recruited subjects from a random sample of board-certified internal medicine and pulmonary specialists, internal medicine and allergy/immunology specialists, and family practice specialists. The present study eligibility is based on follow-up interviews conducted between July 1998 and December 1999, which included 402 subjects.

Address correspondence to M.D. Eisner, Division of Occupational and Environmental Medicine and Division of Pulmonary and Critical Care Medicine, University of California, San Francisco, 350 Parnassus Avenue, Suite 609, San Francisco, CA 94117 USA. Telephone: (415) 476-7351. Fax: (415) 476-6426. E-mail: eisner@itsa.ucsf.edu

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We included the newly developed ETS exposure survey instrument in structured telephone interviews. Subjects with no current personal tobacco smoking and a positive answer to any screening question indicating potential ETS exposure were eligible for the validation study ($n = 235$). The screening questions assessed residence with a smoker and ETS exposure during the previous 7 days at home, another person's home, work, car or other vehicle, bars and nightclubs, and other locations. We enrolled 50 sequential subjects who agreed to participate in the validation study, which included wearing the personal nicotine badge monitor for 7 days and then completing a second telephone administration of the ETS survey instrument. Structured telephone interview content also included demographic characteristics, asthma history, asthma-specific severity and quality of life (24–26), environmental exposures, health status (SF-12) (27), and health care utilization for asthma.

As shown in Table 1, there were no statistical differences in demographic characteristics or smoking status between participants ($n = 50$) and nonparticipants ($n = 185$) who had indicated potential ETS exposure at the initial interview. Compared to nonparticipants, participants had somewhat worse SF-12 physical component summary scores (41.7 vs. 46.5, $p = 0.007$) and severity-of-asthma scores (10.7 vs. 9.2, $p = 0.09$). Conversely, SF-12 mental component summary scores were slightly better among participants (47.2 vs. 44.6, $p = 0.04$). There were no statistical differences in asthma-specific quality-of-life scores. At the initial interview, self-reported duration of ETS exposure during the previous 7 days was similar for participants and nonparticipants ($p = 0.33$). Because of study logistics, the median interval between initial interview

and subsequent validation interview was 225 days (25th–75th interquartile range 138–311 days).

ETS exposure survey instrument. We designed a survey instrument to measure recent ETS exposure among adults with asthma living in northern California (survey included in Appendix). The instrument assesses exposure during the previous 7 days in six microenvironments: the respondent's home, another person's home, traveling in a car or another vehicle, workplace, bars and nightclubs, and other locations. For home exposure, the survey assesses both residence with a smoker and duration of exposure during the previous 7 days (in hours). For each microenvironment, a screening question elicits exposure during the previous 7 days. If there was no exposure, the remaining location-specific items are skipped. Given the workplace smoking restrictions in California, the instrument specifically ascertains exposure in the most likely work-related locations: outdoor smoking areas at work and outdoor job duties. In each area, the instrument ascertains the total duration (in hours) of exposure during the previous 7 days and the intensity of exposure, as indicated by the presence of tobacco smoke odor or visible smoke. In each location, we also assessed exposure-related sensory irritation symptoms (eye and nose irritation) and potential asthma-related respiratory symptoms (coughing, wheezing, or chest tightness). Furthermore, the instrument ascertains whether the subject used extra metered-dose inhaler asthma medications after ETS exposure during the same period.

Personal nicotine badge monitors. Each subject was instructed to wear the personal nicotine badge monitor during regular activities for 7 days. The passive monitor, which has been described previously

(19,20), samples nicotine from ambient air. A 4-cm diameter polystyrene cassette holds a filter treated with sodium bisulfate and a membrane filter functions as a windscreen. Ambient nicotine diffuses to the treated filter, where it is trapped. The collected nicotine is analyzed by gas chromatography with nitrogen selective detection. From the amount of nicotine measured on the filter (micrograms), which represents the total amount of nicotine collected during the monitoring period, we calculated the nicotine concentrations by dividing the nicotine collected by the estimated volume of air sampled (monitoring duration multiplied by sampling rate of 24 mL/min). The passive monitors have a limit of detection $< 0.01 \mu\text{g}$ per filter and a coefficient of variability of 0.11 for replicate analysis (20). We included two control badges (field blanks), which revealed no detectable nicotine.

Statistical analysis. We analyzed the data using SAS version 6.12 (SAS Institute, Cary, NC). We performed bivariate analysis using the chi-square test for categorical variables, t -test for continuous normally distributed variables, and Wilcoxon rank-sum test for non-normally distributed, continuous variables. Because self-reported duration of ETS exposure and measured nicotine concentration were non-normally distributed, we report the median values, range, and interquartile range.

To validate the ETS exposure instrument, we calculated the Spearman rank correlation between total self-reported hours of exposure in all locations with the nicotine concentration measured by personal badge monitoring. In addition to this primary analysis, we tested two alternative scoring systems defined *a priori*. First, we calculated an ETS exposure score weighted for exposure intensity. For each location of

Table 1. Baseline characteristics of subjects participating in personal nicotine badge monitor study compared with other subjects from the asthma cohort.

Variable	Subject group			p -Value for participant vs. nonparticipant
	No ETS exposure ($n = 167$)	ETS exposure, nonparticipant ($n = 185$) ^a	ETS exposure, badge participant ($n = 50$)	
Demographic characteristics				
Age (mean years, SD)	45.0 (7.4)	43.1 (7.8)	44.4 (7.9)	0.30
Sex (% female)	121 (72)	128 (69)	36 (72)	0.70
Race/ethnicity (% white, non-Hispanic)	133 (80)	120 (65)	36 (72)	0.34
Income (median \$, 25th–75th IQR)	62,500 (35,000–87,500)	45,000 (35,000–87,500)	62,500 (31,250–87,500)	0.95
Ever smoked cigarettes (%)	70 (42)	63 (34)	15 (30)	0.59
Currently smoke cigarettes (%)	27 (16)	0	0	—
Asthma severity/health status				
Severity-of-asthma score	9.2 (5.3)	9.2 (5.1)	10.7 (6.8)	0.09
Asthma quality-of-life score	15.3 (14.9)	16.3 (13.8)	18.8 (16.0)	0.27
SF-36 physical summary score	45.8 (12.1)	46.5 (10.7)	41.7 (12.2)	0.007
SF-36 mental summary score	45.4 (7.7)	44.6 (8.3)	47.2 (7.2)	0.04
ETS exposure				
Duration of exposure, past 7 days (median, 25th–75th IQR) ^b	0 (0–0)	0 (0–1)	0 (0–2)	0.33

IQR, interquartile range. All data are mean (SD) unless otherwise indicated.

^aA positive answer to a screening question for each location: home, other person's home, work, car or vehicle, bar or nightclub, or other location. ^bBased on initial interview.

exposure, we weighted exposure duration by 130% for reported tobacco smoke odor and 150% for visible smoke. Second, we calculated an ETS exposure score weighted for sensory irritation and respiratory symptoms (1 point added for each symptom).

To evaluate exposure stability over time, we compared the self-reported duration of ETS exposure during the previous 7 days at the initial and validation telephone interviews. Because duration of exposure was not normally distributed, we performed a rank transformation and then used analysis of variance to calculate the intraclass correlation.

To validate the questions assessing self-reported ETS-attributed symptoms and extra asthma medication use, we used logistic regression analysis to examine the cross-sectional relation between personal nicotine exposure and the risk of sensory irritation symptoms, respiratory symptoms, and extra inhaled asthma medication use. We created indicator variables to reflect lower and higher levels of exposure, which we divided at the median nicotine concentration for subjects with measurable exposure. We tested linear exposure–response relationships by creating an ordinal predictor variable coding the three nicotine concentration categories (none, lower, and higher). We also tested quadratic exposure–response relationships.

Table 2. Prevalence of self-reported ETS exposure among 50 subjects in ETS badge study.

Location	Number	Prevalence (%)
No exposure	12	24
Any exposure	38	76
Indoor/outdoor		
None	12	24
Indoor ^a	20	40
Outdoor only	18	36
Work/home		
None	12	24
Work only	19	38
Home only	5	10
Both home and work	2	4
Other only	12	24

Based on questionnaire administered after monitoring period (validation interview).

^aIncludes 12 subjects with both indoor and outdoor exposure.

Results

ETS exposure and measured nicotine concentration. At the initial interview, 45 of 50 participants reported at least 1 hr of ETS exposure during the previous 7 days, and five additional subjects indicated potential ETS exposure, indicated by residence with a smoker. At the validation interview performed at completion of personal nicotine monitoring, most subjects who initially reported exposure or potential exposure indicated a least 1 hr of ETS exposure (38 of 50, 76%; Table 2). A greater proportion of subjects who reported > 1 hr of ETS exposure at initial interview reported recent exposure at validation interview (37 of 45, 82%).

Of subjects reporting exposure at validation interview, the proportion with indoor exposure and outdoor exposure were comparable (53% and 47%). Of the 20 subjects with indoor exposure, 12 persons also indicated outdoor exposure (60%). The workplace was the major site of exposure (38%), followed by “other” locations, which were all outdoors, and home (14%). All subjects with workplace exposure reported recent exposure in an outdoor smoking area at work, whereas only one subject also reported outdoor exposure during work duties. Only a minority of subjects who lived with a smoker reported any domestic ETS exposure during the previous 7 days (6 of 17 persons, 35%). Conversely, nearly all subjects who had no smoking cohabitants indicated no recent home ETS exposure (32 of 33 persons, 97%).

Table 3 shows the proportion of subjects exposed in each of six microenvironments evaluated by the questionnaire instrument and self-reported exposure duration. Exposure duration ranged from 0 to 70 hr during the previous 7 days. Based on the upper range boundary, bars or nightclubs (55 hr) and the home (50 hr) were the sites associated with greatest maximal self-reported exposure in the previous 7 days.

As measured by the personal nicotine badge monitors, the overall median 7-day nicotine concentration was 0.03 $\mu\text{g}/\text{m}^3$ (25th–75th interquartile range 0–3.69

$\mu\text{g}/\text{m}^3$; Table 4). The measured nicotine concentration varied significantly by self-reported exposure location. Compared with subjects indicating no ETS exposure (median 0 $\mu\text{g}/\text{m}^3$), persons with outdoor exposure (median 0.03 $\mu\text{g}/\text{m}^3$) and indoor exposure (0.05 $\mu\text{g}/\text{m}^3$) had progressively higher levels ($p = 0.03$; Table 4). Of note, more than half of subjects who reported outdoor exposure only had detectable nicotine levels. Measured nicotine concentrations were highest among persons who reported home exposure (median 0.61 $\mu\text{g}/\text{m}^3$), followed by work exposure (0.03 $\mu\text{g}/\text{m}^3$), other (outdoor) exposure (0.025 $\mu\text{g}/\text{m}^3$), and no exposure (0 $\mu\text{g}/\text{m}^3$; $p = 0.03$).

Survey instrument validity. To assess validity, we compared the measurements of ETS exposure based on the survey instrument and the passive badge monitors (Table 5). The Spearman rank correlation coefficient between self-reported ETS exposure duration and directly measured personal nicotine concentration during the same 7-day period was 0.47, indicating moderate agreement ($p = 0.0006$). The exposure scores adjusting for exposure intensity and exposure intensity plus sensory irritation and respiratory symptoms did not improve the correlation (Table 5). Excluding subjects with no measurable nicotine concentration did not affect these results appreciably. To examine the stability of recent ETS exposure over time, we compared the self-reported total hours of ETS exposure ascertained during the validation interview with the initial interview. The intraclass correlation was 0.72, indicating moderate to high stability over time.

ETS exposure and the risk of symptoms.

To evaluate the validity of self-reported ETS-attributed symptoms, we examined the cross-sectional relation between directly measured personal nicotine concentration and the risk of sensory irritation symptoms, respiratory symptoms, and medication use. Compared to persons with no measured exposure, lower-level and higher-level ETS exposures were associated with increased risk of sensory symptoms, including eye, nose,

Table 3. ETS exposure by location among 50 adults with asthma.

Location	No. exposed during past 7 days (%)	Exposure duration during past 7 days (hr)				Exposure intensity among exposed subjects	
		Minimum	Median	Mean	Maximum	No. who smelled smoke (%)	No. who saw smoke (%)
Home	7 (14)	0	0	2.3	50	6 (86)	6 (86)
Other person's home	8 (16)	0	0	0.3	8	8 (89)	3 (33)
Car or vehicle	6 (12)	0	0	0.5	20	N/A	N/A
Work—outdoor smoking area ^a	21 (42)	0	3	5.5	40	17 (63)	N/A
Bar or nightclub	5 (10)	0	0	1.3	55	N/A	3 (60)
Other ^b	12 (24)	N/A	N/A	N/A	N/A	N/A	N/A
Total	38 (76)	0	0	4.6	70	24 (48)	10 (20)

N/A, not asked.

^aNumber of times walked through smoking area (not hours of exposure). ^bAll were outdoor sites.

and throat irritation [odds ratio (OR) 2.2 and 5.9, respectively; Table 6]. Lower- and higher-level ETS exposures also were related to an increased risk of respiratory symptoms (OR 1.9 and 6.8) and extra bronchodilator use after ETS exposure (OR 2.2 and 8.1). For all three outcomes, there was statistical evidence of a simple exposure–response relation ($p = 0.054$, 0.017 , and 0.022 , respectively). There was no evidence of a quadratic exposure–response relation ($p > 0.5$ in all cases).

Discussion

We developed an ETS exposure instrument that is brief and easy to administer by telephone interview. The ETS questionnaire can be incorporated easily into a larger survey instrument. Self-reported total hours of ETS exposure during the previous 7 days correlated moderately with measured nicotine concentration, supporting the instrument's validity. More complex scoring systems that incorporate intensity of exposure or intensity plus respiratory and sensory irritation symptoms do not appreciably

improve instrument validity. Exposure monitoring also supported the validity of self-reported ETS-attributed symptoms and medication use. ETS exposure, measured by the personal nicotine badge, was associated with a greater risk of ETS-attributed sensory irritation symptoms, respiratory symptoms, and extra medication use.

We found a moderate correlation between self-reported ETS exposure duration and direct personal nicotine measurements, consistent with previous studies that used cotinine as a biomarker (7–16). Fewer studies have used personal nicotine monitoring, which measures actual exposure, to validate questionnaire instruments. In a study of 415 pregnant women, O'Connor and colleagues (28) demonstrated a similar correlation between total duration of ETS exposure and nicotine concentration ($r = 0.41$). The investigators cautioned that their results should not be extrapolated to nonpregnant women, who may have different patterns of exposure. Other investigators used a more complicated questionnaire instrument that

assessed duration, intensity, and proximity of each individual ETS exposure during the previous week (29). Using personal nicotine monitoring, they found a higher correlation with an index of ETS exposure based on these variables ($r = 0.91$). This questionnaire requires subject response entry in multicell tables that would be difficult to administer by telephone.

For most subjects, the observed nicotine concentrations were low, with a median of $0.03 \mu\text{g}/\text{m}^3$ (range $0\text{--}3.69 \mu\text{g}/\text{m}^3$). Compared to our study, Coghlin and colleagues (29) found higher nicotine concentrations among 53 volunteers who wore personal monitors during a typical week (median $1.7 \mu\text{g}/\text{m}^3$ and $2.8 \mu\text{g}/\text{m}^3$ in two different periods) (29). The observed concentrations among 50 adults with asthma were more comparable to those of pregnant women who reported ETS exposure (median $0.1 \mu\text{g}/\text{m}^3$). Pregnant women who reported only social exposure had even lower concentrations (median $0.08 \mu\text{g}/\text{m}^3$). Both pregnant women and adults with asthma, because of their health conditions, might be expected to limit their ETS exposure.

Adults with asthma living in northern California had low levels of ETS exposure, reflecting the restrictions on public smoking. Moreover, the locations where ETS exposure occurs appear to be changing. Residence with a smoker, which has been used in many epidemiologic studies as a key marker of ETS exposure (30–32), was a poor indicator of recent exposure. Only a minority of adults with asthma who live with a smoker reported any exposure during the previous 7 days, most likely because smoking was not permitted indoors. A shift from indoor to outdoor exposure was also observed, with 38% of subjects indicating outdoor workplace exposure. Future ETS exposure survey instrument should take these temporal trends into account.

This validation study is potentially limited by the low observed exposure levels, which may have attenuated the correlation between self-reported exposure and nicotine concentration. However, these low exposure levels reflect current restrictions in public smoking that require consideration in epidemiologic studies. Even among persons who may have lower than average ETS exposure due to their disease, the instrument appears to have adequate validity. The ETS exposure survey instrument also measures a 7-day period, which may not be fully representative of a subject's usual exposure. Furthermore, our instrument, which is adapted for use in California, does not assess indoor workplace exposure. A

Table 4. Location of ETS exposure and measured nicotine exposure level.

Location	No. of subjects	Nicotine concentration ($\mu\text{g}/\text{m}^3$)			p -Value ^a
		Median	Range	25th–75th IQR	
Indoor/outdoor					0.03
None	12	0	0–0.06	0–0.035	
Outdoor only ^b	18	0.03	0–0.08	0–0.05	
Indoor	20	0.05	0–3.69	0–0.49	
Home/work					0.03
None	12	0	0–0.06	0–0.035	
Work only	19	0.03	0–0.36	0–0.07	
Home ^c	7	0.61	0–3.69	0.02–3.03	
Other location only	12	0.025	0–0.84	0–0.05	
Total	50	0.03	0–3.69	0.03–0.06	

IQR, interquartile range.

^aKruskal-Wallis test for comparison of nicotine concentration between groups. ^bSixty-one percent ($n = 11/18$) of subjects who reported outdoor exposure only had detectable nicotine levels. ^cIncludes two subjects with both home and work ETS exposure.

Table 5. Validity of questionnaire measurement of ETS exposure.

Questionnaire-based measure	Spearman correlation with nicotine concentration	p -Value
Exposure duration (hours/previous 7 days)	0.47	0.0006
Score adjusted for exposure intensity ^a	0.46	0.0007
Score adjusted for exposure intensity, sensory irritation symptoms, and respiratory symptoms ^b	0.45	0.001

^aScore adjusted for exposure intensity had median 1.6, range 0–128, 25th–75th interquartile range 0.7–3.2. ^bScore adjusted for exposure intensity, sensory irritation symptoms, and respiratory symptoms had median 1.9, range 0–137, 25th–75th interquartile range 0.8–5.0.

Table 6. ETS exposure (nicotine level) and the risk of sensory irritation symptoms, respiratory symptoms, and extra bronchodilator use.

Measured nicotine level	n	Sensory irritation (OR, 95% CI)	Respiratory symptoms (OR, 95% CI)	Extra bronchodilator use (OR, 95% CI)
None (referent)	21	1.0	1.0	1.0
Lower level ($0\text{--}0.05 \mu\text{g}/\text{m}^3$)	16	2.2 (0.3–15)	1.9 (0.4–8.8)	2.2 (0.3–15)
Higher level ($> 0.05 \mu\text{g}/\text{m}^3$)	13	5.9 (0.95–37)	6.8 (1.4–32.3)	8.1 (1.3–50)
p -Value for trend		0.054	0.017	0.022

*Sensory irritation symptoms: eye or nose irritation; respiratory symptoms: cough, wheezing, or dyspnea.

section for indoor work exposure can be easily added, modeled on those developed for other microenvironments (available from the authors). Further research will be necessary to evaluate the survey in other environmental settings, such as large industrial workplaces, where building ventilation may be different. In such settings, tailor-made survey instruments could be required.

Validation of the ETS exposure survey instrument was also limited by measurement of a single marker of ETS exposure. Because ETS contains more than 4,000 chemical compounds, it is not feasible to measure all ETS constituents (32,33). Although nicotine is a highly specific marker of ETS, it may not always track with the ETS components responsible for disease causation or exacerbation. In particular, the relationship between nicotine concentration and other ETS constituents may be affected by factors such as building ventilation rate and indoor air volume (17,33). In addition, nicotine may adsorb to indoor surfaces and be reemitted in the absence of active smoking, changing its concentration relative to other ETS constituents (17). Despite these factors, field studies indicate that nicotine correlates strongly with other ETS constituents, including respiratory suspended particles (34,35) and 3-ethenylpyridine (a volatile organic compound unique to ETS) (34).

Most epidemiologic studies of ETS-related health effects have used self-reported ETS exposure. All survey-based methods of ascertaining ETS exposure are potentially limited by information bias (i.e., misclassification of exposure). For example, persons with symptomatic respiratory disease may be more likely to remember and report recent ETS exposure. In our study of adults with asthma, personal nicotine badge monitoring was a feasible method for directly measuring ETS exposure, potentially minimizing this information bias.

A brief survey instrument can be used to assess ETS exposure among adults with asthma, even with low levels of exposure. The instrument is valid and can be incorporated into larger telephone-based interviews. This instrument could be a valuable tool for studying the effect of ETS exposure on adult asthma health outcomes.

REFERENCES AND NOTES

- Ostro BD, Lipsett MJ, Mann JK, Wiener MB, Selner J. Indoor air pollution and asthma. Results from a panel study. *Am J Respir Crit Care Med* 149:1400–1406 (1994).
- Sippel JM, Pedula KL, Vollmer WM, Buist AS, Osborne ML. Associations of smoking with hospital-based care and quality of life in patients with obstructive airway disease. *Chest* 115:691–696 (1999).
- Jindal SK, Gupta D, Singh A. Indices of morbidity and

Appendix

Environmental Tobacco Smoke Questionnaire Instrument

The next series of questions ask about your exposure to other people's tobacco smoke.

Q1. Many people have different approaches to tobacco smoking in their household. Which is the best description of tobacco smoking in your home? Would you say that:

- (1) You never allow smoking inside your home,
- (2) Smoking is allowed only in certain rooms, but not in other areas of your living space, or
- (3) Smoking is allowed in all rooms of your home.

Q2. Do you live in the same household with someone who smokes tobacco products?

[If yes to Q2] Q3. How many people in your household smoke?

[If Q1 = 2 or 3 and Q2 = yes] Q4. Has anyone smoked tobacco in your home in the past seven days?

[If yes to Q4, then ask next series; else skip to Q12]

Q5. In the past 7 days, how many hours in total were you exposed to someone else's tobacco smoke at home?

Q6. During the past 7 days, did you enter a room in your home that was visibly smoky?

Q7. In the past 7 days, did you smell tobacco smoke in your home?

You said that you were exposed to someone else's tobacco smoke in your home. During the past 7 days, did you experience any of the following after this exposure:

Q8. Red eyes or eye irritation?

Q9. Runny nose or nose irritation?

Q10. Coughing, wheezing, or chest tightness?

Q11. In the past 7 days, did you take extra asthma sprays after exposure to tobacco smoke in your home?

Q12. In the past 7 days, have you visited another person's home where someone was smoking tobacco products indoors?

[If yes to Q12 then ask next series; else skip to Q20]

Q13. In the past 7 days, how many hours in total were you exposed to someone else's tobacco smoke in another person's home?

Q14. During the past 7 days, did you enter a room in another person's home that was visibly smoky?

Q15. In the past 7 days, did you smell tobacco smoke in another person's home?

You answered that you were exposed to someone else's tobacco smoke in another person's home. During the past 7 days, did you experience any of the following after this exposure:

Q16. Red eyes or eye irritation?

Q17. Runny nose or nose irritation?

Q18. Coughing, wheezing, or chest tightness?

Q19. In the past 7 days, did you take extra asthma sprays after exposure to tobacco smoke in another person's home?

Q20. In the past 7 days, have you traveled by car or other vehicle with someone else who was smoking tobacco products?

[If yes to Q20 then ask next series; else skip to Q26]

Q21. In the past 7 days, how many hours in total did you spend traveling in a car while someone was smoking tobacco?

You answered that you were exposed to someone else's tobacco smoke while traveling by car. During the past 7 days, did you experience any of the following after this exposure:

Q22. Red eyes or eye irritation?

Q23. Runny nose or nose irritation?

Q24. Coughing, wheezing, or chest tightness?

Q25. In the past 7 days, did you take extra asthma sprays after exposure to someone else's tobacco smoke in a car?

Q26. Is there an outdoor area at your workplace where cigarette smokers routinely gather or congregate in order to smoke?

[If yes to Q26 then ask next series; else skip to Q33]

Q27. In the past 7 days, how many times did you walk through or past this area while others were smoking?

Q28. While walking through or past this area, did you smell smoke?

In the past 7 days, did walking through or past this area result in your experiencing any of the following:

Q29. Red eyes or eye irritation?

Q30. Runny nose or nose irritation?

Q31. Coughing, wheezing, or chest tightness?

Q32. In the past 7 days, did you take extra asthma sprays after walking through or past this area?

Q33. Do your job duties directly involve working outdoors one or more hours per week?

[If yes to Q33] Q34. During your work outdoors, do any of your coworkers smoke tobacco products in your presence?

[If yes to Q34 then ask next series; else skip to Q42]

Q35. In the past 7 days, how many hours did you spend near coworkers who were smoking tobacco outdoors?

Q36. During the past 7 days, did you smell tobacco smoke while working outdoors?

You answered that you were exposed to your coworkers' tobacco smoke while working outdoors. During the past 7 days, did you experience any of the following after this exposure:

Q37. Red eyes or eye irritation?

Q38. Runny nose or nose irritation?

Q39. Coughing, wheezing, or chest tightness?

Q40. In the past 7 days, did you take extra asthma sprays after exposure to tobacco smoke outdoors?

Q41. In the past 7 days, did you take extra asthma sprays after working outdoors WITHOUT tobacco smoke exposure?

Q42. In the past 7 days or nights, were you in a bar, nightclub, cocktail lounge, sports arena, or concert hall where someone else was smoking tobacco products?

[If yes to Q42 then ask next series; else skip to Q50]

Q43. In the past 7 days, how many hours in total were you exposed to someone else's tobacco smoke in a bar or other place of entertainment?

Q44. During the past 7 days, did you enter a room in a bar or other place of entertainment that was visibly smoky?

Q45. In the past 7 days did your clothes smell like tobacco smoke after returning home from a bar or other place of entertainment?

You answered that you were exposed to someone else's tobacco smoke in a bar or other place of entertainment.

During the past 7 days, did you experience any of the following after this exposure:

Q46. Red eyes or eye irritation?

Q47. Runny nose or nose irritation?

Q48. Coughing, wheezing, or chest tightness?

Q49. In the past 7 days, did you take extra asthma sprays after exposure to tobacco smoke in a bar or other place of entertainment?

Q50. I have asked you about exposure to someone else's tobacco smoke in your home, friends' homes, work, and bars or nightclubs. In the past 7 days, was there any other location where you were exposed to tobacco smoke?

[If yes to Q51 then ask next series; else skip to end]

Q51. Specify: _____

During the past 7 days, did you experience any of the following after this exposure:

Q52. Red eyes or eye irritation?

Q53. Runny nose or nose irritation?

Q54. Coughing, wheezing, or chest tightness?

Q55. Did you use extra asthma sprays after exposure to someone else's tobacco smoke in this place?

Administration and Scoring

Unless otherwise specified, all questions have yes/no response options. For each microenvironment, a screening question (in bold font) ascertains whether any ETS exposure occurred during the past 7 days. If the subject indicates ETS exposure in that environment, then a series of questions assess duration of exposure, exposure intensity, and symptoms/asthma medication use. Otherwise, the survey skips to the next microenvironment screening question.

To determine the total duration of exposure during the past 7 days, sum the individual microenvironment exposure durations (Q5 + Q13 + Q21 + Q35 + Q43). An ETS exposure score weighted for exposure intensity can also be calculated. For each location of exposure, we weighted exposure duration by 130% for reported tobacco smoke odor and 150% for visible smoke. A second ETS exposure score weighted for sensory irritation and respiratory symptoms is calculated by further adding 1 point added for each symptom.

control of asthma in adult patients exposed to environmental tobacco smoke. *Chest* 106:746–749 (1994).

4. Blanc PD, Ellbjär S, Janson C, Norback D, Norrman E, Plaschke P, Toren K. Asthma-related work disability in Sweden. *Am J Respir Crit Care Med* 160:2028–2033 (1999).
5. State-specific prevalence of current cigarette and cigar smoking among adults—United States, 1998. *Morb Mortal Wkly Rep* 48:1034–1039 (1999).
6. Macdonald HR, Glantz SA. Political realities of statewide smoking legislation: the passage of California's Assembly Bill 13. *Tob Control* 6:41–54 (1997).
7. Delfino RJ, Ernst P, Jaakkola MS, Solomon S, Becklake MR. Questionnaire assessments of recent exposure to environmental tobacco smoke in relation to salivary cotinine. *Eur Respir J* 6:1104–1108 (1993).
8. Kemmeren JM, van Poppel G, Verhoef P, Jarvis MJ. Plasma cotinine: stability in smokers and validation of self-reported smoke exposure in nonsmokers. *Environ Res* 66:235–243 (1994).
9. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the U.S. population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988 to 1991. *JAMA* 275:1233–1240 (1996).
10. Rebagliato M, Bolumar F, Florey CD. Assessment of exposure to environmental tobacco smoke in nonsmoking pregnant women in different environments of daily living. *Am J Epidemiol* 142:525–530 (1995).
11. Emmons KM, Abrams DB, Marshall RJ, Etzel RA, Novotny TE, Marcus BH, Kane ME. Exposure to environmental tobacco smoke in naturalistic settings. *Am J Public Health* 82:24–28 (1992).
12. Emmons KM, Abrams DB, Marshall R, Marcus BH, Kane M, Novotny TE, Etzel RA. An evaluation of the relationship between self-report and biochemical measures of environmental tobacco smoke exposure. *Prev Med* 23:35–39 (1994).
13. Riboli E, Preston-Martin S, Saracci R, Haley NJ, Trichopoulos D, Becher H, Burch JD, Fonham ET, Gao YT, Jindal SK, et al. Exposure of nonsmoking women to environmental tobacco smoke: a 10-country collaborative study. *Cancer Causes Control* 1:243–252 (1990).
14. Coultas DB, Peake GT, Samet JM. Questionnaire assessment of lifetime and recent exposure to environmental tobacco smoke. *Am J Epidemiol* 130:338–347 (1989).
15. Masi MA, Hanley JA, Ernst P, Becklake MR. Environmental exposure to tobacco smoke and lung function in young adults. *Am Rev Respir Dis* 138:296–299 (1988).
16. Jaakkola MS, Jaakkola JJ, Becklake MR, Ernst P. Passive smoking and evolution of lung function in young adults. An 8-year longitudinal study. *J Clin Epidemiol* 48:317–327 (1995).
17. Jaakkola MS, Jaakkola JJ. Assessment of exposure to environmental tobacco smoke. *Eur Respir J* 10:2384–2397 (1997).
18. Benowitz NL. Biomarkers of environmental tobacco smoke exposure. *Environ Health Perspect* 107(suppl 2):349–355 (1999).
19. Hammond SK, Sorensen G, Youngstrom R, Ockene JK. Occupational exposure to environmental tobacco smoke. *JAMA* 274:956–960 (1995).
20. Hammond SK, Leaderer B. A diffusion monitor to measure exposure to passive smoking. *Environ Sci Tech* 21:494–497 (1987).
21. Blanc PD, Cisternas M, Smith S, Yelin EH. Asthma, employment status, and disability among adults treated by pulmonary and allergy specialists. *Chest* 109:688–696 (1996).
22. Blanc PD, Eisner MD, Israel L, Yelin EH. The association between occupation and asthma in general medical practice. *Chest* 115:1259–1264 (1999).
23. Blanc P. Correction to the scientific record [Letter]. *Chest* 118:564 (2000).
24. Eisner MD. Erratum to Assessment of asthma severity in adults with asthma treated by family practitioners, allergists, and pulmonologists. *Med Care* 38:880–885 (2000).
25. Eisner MD, Katz PP, Yelin EH, Henke J, Smith S, Blanc PD. Assessment of asthma severity in adults with asthma treated by family practitioners, allergists, and pulmonologists. *Med Care* 36:1567–1577 (1998).
26. Marks GB, Dunn SM, Woolcock AJ. A scale for the measurement of quality of life in adults with asthma. *J Clin Epidemiol* 45:461–472 (1992).
27. Ware J Jr, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care* 34:220–233 (1996).
28. O'Connor TZ, Holford TR, Leaderer BP, Hammond SK, Bracken MB. Measurement of exposure to environmental tobacco smoke in pregnant women. *Am J Epidemiol* 142:1315–1321 (1995).
29. Coghlin J, Hammond SK, Gann PH. Development of epidemiologic tools for measuring environmental tobacco smoke exposure. *Am J Epidemiol* 130:696–704 (1989).
30. Wells AJ. Passive smoking as a cause of heart disease. *J Am Coll Cardiol* 24:546–554 (1994).
31. Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ* 315:980–988 (1997).
32. California EPA. Health Effects of Exposure to Environmental Tobacco Smoke. Sacramento, CA:California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, 1997.
33. Daisey JM. Tracers for assessing exposure to environmental tobacco smoke: what are they tracing? *Environ Health Perspect* 107(suppl 2):319–327 (1999).
34. Jenkins RA, Palausky A, Counts RW, Bayne CK, Dindal AB, Guerin MR. Exposure to environmental tobacco smoke in sixteen cities in the United States as determined by personal breathing zone air sampling. *J Expo Anal Environ Epidemiol* 6:473–502 (1996).
35. Leaderer BP, Hammond SK. Evaluation of vapor-phase nicotine and respirable suspended particle mass as markers for environmental tobacco smoke. *Environ Sci Technol* 25:770–777 (1991).